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worked for six hours with his hands in a six per cent cresylic acid solution; the next day a severe exfoliative dermatitis of the hands, and a right facial paralysis began, progressed in severity for several days, and then regressed so that complete recovery is expected. The exfoliation of skin exposed to the acid probably represents the direct action of a cytoplasmic poison; the relation of the drug to the facial paralysis is not clear, and may be accidental, but the occurrence points to the need for caution in the use of cresylic acid as a solvent.

—B. H. Landing.

TOXIC POLYNEURITIS ARISING DURING THE MANUFACTURE OF TRICRESYL PHOSPHATE. Donald Hunter, Kenneth M. A. Perry and R. Byron Evans. *Brit. J. Indust. Med.*, vol. 1, no. 4, pp. 227-231 (Oct., 1944).

The literature of the toxicity of tricresyl and triphenyl phosphate is reviewed. An account is given of the cases of three men who developed a toxic polyneuritis while working in industry in the manufacture of tricresyl phosphate. Experiments on domestic fowls confirm that tri-ortho-cresyl phosphate is toxic. 3.5 g. of tri-ortho-cresyl phosphate killed two twelve-week old cockerels. 12.5 g. of tri-meta-cresyl phosphate produced weakness of the legs, and 15.5 g. respiratory paralysis, whereas 25 g. of tri-para-cresyl phosphate and triphenyl phosphate produced no effect. Measures for the prevention of poisoning are discussed.—Authors' summary.

GYNECOMASTIA IN STILBESTROL WORKERS. M. Patricia Fitzsimons. *Brit. J. Indust. Med.*, vol. 1, no. 4, pp. 235-237 (October, 1944).

Twenty cases of gynecomastia occurring among 38 men engaged in the manufacture of stilbestrol are described. An account is given of the preventive measures found necessary to protect the work-people.—Authors' summary.

CHLOROLEUKEMIA AS A RESULT OF BENZOL POISONING. H. Rackner. *Deut. med. Woch.*, vol. 70, no. 15/16, pp. 219-221 (April 14, 1944).

A description of a fatal case of leukemia, with the unusual feature of widespread "green tumor" infiltration, occurring in a man employed for 19 years as a vulcanizer, two years after exposure to benzol (benzene) had practically ceased.

The period between the onset of symptoms of general weakness and lassitude and the fatal termination, with fever, gangrenous tonsillitis, and hemorrhage from mucous membranes and into the pleural cavities, was 10 years. The absence of long-standing blood changes is shown by the results of a blood examination made 10 years earlier while the man was under treatment for renal calculus; the total white cell count was then 100 per cmm.

On admission to hospital the blood count was R.B.C. 1.5 millions; Hb 38 per cent; W.B.C. 26,000; differential

count showed 56 per cent myelocytes with numerous myeloblasts, 28 per cent polymorphs and 14 per cent lymphocytes.

The initial improvement occurring under treatment, which included blood transfusion, was not maintained and death took place 2½ months later.

Post-mortem examination showed infiltration of sternum, ribs, spinal column, parietal pleura, liver and kidneys with masses of greenish tissue consisting of irregular round and oval cells. Lymph glands around the aorta, head of the pancreas, ligamentum gastroduodenale, and the axilla, and the spleen, were enlarged and showed green coloration.

In ascribing the disease to the effect of benzol, the author quotes cases from the literature in which both aplastic anemia and leukemia have occurred after a latent period of non-exposure, and advances Schilling's hypothesis that benzol leukemia is the expression of an over-compensation of the blood-forming tissues following an initial aleukemia effect. Although this particular form of chloroleukemia has not previously been described as a result of exposure to benzol, he believes that benzol poisoning was at least the precipitating exogenous factor in this case.—*Bull. of Hygiene*.

INVESTIGATION FOR SIGNS OF BENZENE INTOXICATION IN WORKERS USING AEROPLANE DOPE AND RUBBER SOLVENTS. Donald Hunter, Reginald Milton, Kenneth M. A. Perry, H. J. Barrie, J. F. Louit and T. S. Marshall. *Brit. J. Indust. Med.*, vol. 1, no. 4, pp. 238-246 (October, 1944).

In this investigation concentration of benzene in the atmosphere of dope shops and factories using rubber solvents did not approach toxic levels. Blood counts of workers in these factories were not significantly different from counts in a control group. No material change was found in the inorganic-total sulphate ratio in the urine of the workers. It is emphasized that, in the absence of estimations of atmosphere concentrations of benzene, blood changes alone cannot be accepted as evidence of benzene intoxication.—Authors' summary.

WELDING FUMES: FACTS AND FANCY. Morwick Ross and Philip Drinker. *Nat. Safety News*, vol. 51, no. 2, pp. 14-15, 80-86 (Feb., 1945).

This article "debunks" the present imaginary ailments attributed to welding fumes. Such notions as coated lungs and falling hair have been blamed on welding fumes without any basis whatsoever. The use of ventilation in ship construction is stressed and its value emphasized. Ventilation improves working conditions and increases production. Methods of ventilating cargo holds, double bottoms, and forepeaks are schematically illustrated. A brief description of the types of blowers used is included. A standard design of ventilation is also described. The latter procedure

1. Hydrated alumina and metallic aluminum both seem to be effective in relieving the symptoms of silicosis in a significant number of cases.

2. A material difference in the effectiveness of the two powders, if any exists, can not be determined from such a limited series of cases.

3. Ordinary clinical methods are inadequate to reveal improvement in work performance following the treatments.

4. The hydrate seems to be superior to the metallic powder for the treatment of silicosis and deserves further clinical trial.—*Author's summary.*

ROCK WOOL NOT CAUSE OF PNEUMOCONIOSIS. J. A. M. A. (*Queries and Minor Notes*), vol. 128, no. 3, p. 244 (May 19, 1945).

Information was requested on a condition apparently caused by inhalation of rock wool and other fine particles passing into the lung proper.

Answer: Information on the chronic pathologic effects of inhaled rock wool is practically nonexistent. Fibers of glass wool are not inhalable, and it is assumed that rock wool is likewise excluded from the lungs because of the size and physical properties of the fiber. X-ray films of persons exposed to this material tend to confirm the inference, for none of those observed in any way resemble the reaction to the known fibrous dust asbestos. If there is any change noted at all it is limited to an exaggeration of the normal linear markings due to particles and broken fragments of the fiber; such changes are rarely associated with symptoms or disability. Diagnosis would depend on a history of exposure over periods measured in years, the demonstration of a uniformly altered X-ray pattern in a considerable group of workmen and a physical examination for evidence of disability. The prognosis should be good, and no disability or increased susceptibility to pulmonary infection is likely. Treatment is entirely prophylactic.—J. A. M. A.

FIBERGLAS" NOT CAUSE OF PNEUMOCONIOSIS. J. A. M. A. (*Queries and Minor Notes*), vol. 128, no. 7, p. 554 (June 16, 1945).

Compartments in the service ships are being lined with mats of "Fiberglas," presumably for insulating and sound proofing purposes. Several shipworkers have had severe persisting cough, sometimes lasting many months, which they ascribe to the "Fiberglas" dust. Some men have physical signs of bronchitis, some do not. One man who had coughed for seven months—since beginning to work with the "Fiberglas"—has no definite physical signs of silicosis, and his X-ray film is normal except for a number of Ghon shadows. Is there not an industrial hazard of silicosis? No respirators or masks are issued.

Answer: Unpublished animal experiments at the Wadsworth Laboratory have demonstrated that "Fiber-

glas" is not inhalable in appreciable quantities. After exposures of three years with the maximum atmospheric concentrations that could be maintained, the lungs of guinea pigs and white rats contained practically no fiber, and no scar tissue developed. The fiber used for insulation varies from 3 to 10 microns in diameter, which is too coarse to penetrate the lungs. A specially prepared fiber, 3 microns or less in diameter, was also excluded from the lungs apparently because it carries an electrostatic charge which causes it to adhere and mat on surfaces with which it comes in contact. Annual X-ray examinations of employees manufacturing this product have revealed no signs of pulmonary reaction. While Fiberglas is irritating to the skin and upper respiratory membranes, it does not cause pneumoconiosis.—J. A. M. A.

THE ROENTGENOGRAM AND SOME CHRONIC NON-TUBERCULOUS PULMONARY CONDITIONS. A. Rest. *Am. J. Roentgenol.*, vol. 51, pp. 434-438 (Apr., 1944).

Cases of various lung conditions are presented. In tuberculosis case finding, the roentgen ray plays a great part in the diagnosis of that disease. However, a plea is made not to be too hasty in making the diagnosis of pulmonary tuberculosis on roentgen findings alone without consideration of other laboratory procedures. It cannot be emphasized too strongly that when a patient expectorates sputum which originates from below the bifurcation of the trachea, that sputum should contain tubercle bacilli if an open case of tuberculosis exists. The failure to find tubercle bacilli in the sputum should result in an investigation for non-tuberculous pulmonary disease as shown in my series of cases.—*Author's summary.* (In the list of cases, only one of calcicosis was of possible industrial origin. The patient had worked for 11 years as a floor man in an embroidery concern, which brought him into prolonged inhalation of fine chalk dust used as a "stamping powder." Although dusts rich in calcium are considered the least harmful of organic dusts, their presence in the lungs give a characteristic picture and symptoms without serious effect on health.)—*Ind. Hyg. Found.*

AN OUTLINE OF RECENT ADVANCES IN THE EXPERIMENTAL PRODUCTION OF TUMORS. R. A. Willis. *Med. J. Australia*, vol. 1, no. 15, pp. 361-368 (April 14, 1945).

The author reviews the experimental work done in the production of neoplasms. The production of sarcomata and carcinomas by some of the same agents suggests that neoplastic change is fundamentally similar in all tissues. Thus an advance in the knowledge of the nature of the change in one type of tissue will probably shed light on the entire cancer problem.

As the latent period between the carcinogenic stimulus and the appearance of the tumor may be from

SYSTEMIC AND INFECTIOUS OCCUPATIONAL DISEASES; AND HAZARDS

SOME NEW ASPECTS AND APPROACHES TO THE PROBLEM OF DUST DISEASES. *Carl U. Dernehl and Carl A. Nau. Texas Reports on Biology and Medicine, vol. 3, no. 2, pp. 227-237 (Summer, 1945).*

Increasing industrialization has led to widespread exposure to many dusts, natural and synthetic. The authors present current theories regarding pathogenesis and treatment of several important dust diseases and describe a new experimental tool for further investigation of dust diseases. Pulmonary fibrosis in response to silicon dioxide dust is a serious problem for which a hydrated form of aluminum offers high promise as an inhalant agent in prevention and in partial therapy. Cotton dust is capable of producing pulmonary changes that are not the fibrotic proliferation of silicosis but rather a diffuse, fine increase in the alveolar connective tissue framework. A sensitivity to cotton dust protein is necessary to produce an interstitial pneumonitis, but chronic irritation due to mere presence of cotton dust may over a span of years produce dilatation and emphysema due to repeated coughing. Welders' siderosis appears to be innocuous; the lung markings being due to the presence of iron pigment in the lung. However, new alloy welding rods with special coatings may be less benign; detailed studies are needed. A new apparatus is described for exposing animals to inhalant dust mixtures for long periods so that industrial conditions may be closely paralleled.—*Weldon W. West.*

ROENTGENOLOGY OF THE MASSIVE CONGLOMERATE LESIONS OF SILICOSIS. *Mortimer R. Camiel. Am. Rev. Tuberc., vol. 51, no. 6, pp. 527-531 (June, 1945).*

1. The roentgenological criteria for the diagnosis of a group of conglomerate lesions of silicosis are enumerated.

2. Using these criteria, the correct diagnosis may be suggested from the roentgenographic study alone, even in the absence of a confirmatory occupational exposure.

3. Two cases with suggestive occupational exposure are presented. In addition to the occupational history, exact knowledge of the type of dust inhaled is necessary for the logical establishment of a hazard. The typical roentgenological findings of silicosis in these cases are in accord with the inhalation of free silica.—*Author's summary.*

SUSPECTED PNEUMOCONIOSIS OF GRAPHITE WORKERS. *British Med. J., no. 4408, p. 916 (June 30, 1945).*

A discussion of a report by L. Dunner (*Brit. J. Radiol.*, 1945, 18, 33), of skiagrams of five workers exposed to graphite dust for 17 to 30 years, and showing varying degrees of fibrosis. Three of the men had cough with shortness of breath, and two were without symptoms. All were able to work. It is suggested

that Dunner's presumed lesions may have been only accumulations of graphite.—*Cutland C. Brown, Jr.*

SYSTEMIC SARCOIDOSIS. *J. F. Briggs. Minn. Med., vol. 28, no. 6, pp. 452-454 (June, 1945).*

A brief review of the pathology and symptomatology of Boeck's sarcoidosis is presented. Two case histories are included. This condition should be suspected when a patient seems to have tuberculosis but with negative smears, cultures and skin tests.—*Charles B. Beal.*

ISOLATION AND IDENTIFICATION OF THE CAUSATIVE AGENT IN EPIDEMIC KERATOCONJUNCTIVITIS (SUPERFICIAL PUNCTATE KERATITIS) AND HERPETIC KERATOCONJUNCTIVITIS. *Alfred E. Maumenee, Guy S. Hayes, and Thomas L. Harman. Am. J. Ophthalm., vol. 28, no. 8, pp. 823-839 (August, 1945).*

Herpes-simplex virus can cause a keratoconjunctivitis which is clinically almost identical to that caused by the "E. K." virus. In spite of the similarity in the clinical picture and the cross immunologic reactions of the two viruses, they are thought to be separate entities but are probably of the same genus. Intranuclear inclusion bodies produced by the "E. K." virus have been demonstrated experimentally for the first time. Methylene-blue therapy for herpes and epidemic keratoconjunctivitis appears to be of some value in the early stages of these diseases.—*Authors' summary.*

THE METHYLENE BLUE TEST IN INFECTIOUS (EPIDEMIC) HEPATITIS. *Sydney S. Gellis and Joseph Stokes, Jr. J. A. M. A., vol. 128, no. 11, pp. 782-783 (July 14, 1945).*

Methylene blue tests were performed daily on 77 patients with hepatitis. Of these, 33 showed positive reactions one to six days before scleral icterus appeared. In 12 instances, a positive test was obtained while the icteric index was still normal. The remaining 44 patients showed jaundice on admission. During convalescence, the test was negative, even while the patient was still jaundiced. In the five patients who suffered relapses, the test became positive again. This paper supports the much-discussed work of Clyde P. Myers (*J. Indust. Hyg. & Toxicol.*, 27: 52, 1945).—*Cutland C. Brown, Jr.*

PSEUDO-RAYNAUD'S DISEASE DUE TO ELECTRIC VIBRATORY TOOLS. *T. Brocklehurst. Med. Press & Circular, pp. 10-11 (Jan. 3, 1945).*

A condition resembling the first stage of Raynaud's disease has been observed in people who work with vibratory hand tools. In some cases it has been attri-

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